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=> e gardyn j, 1995/re
E1            1     GARDYN E, 1994, P148, P 1 AUSTR CONC STRUC/RE
E2            1     GARDYN E, 1997, P267, P C INF QUAL CAMBR M/RE
E3            0 --> GARDYN J, 1995/RE
E4            1     GARDYN J, 1995, V50, P292, AM J HAEMATOL/RE
E5           10     GARDYN J, 1995, V50, P292, AM J HEMATOL/RE
E6            3     GARDYN J, 1995, V50, P292, AMERICAN JOURNAL OF HEMATOLOGY/RE
E7            1     GARDYN J, 1995, V50, P2925, AM J HEMATOL/RE
E8            1     GARDYN J, 2001, V74, P76, INT J HEMATOL/RE
E9            1     GARDYN R, 2002, P31, AM DEMOGRAPHICS  MAY/RE
E10           1     GARDYNE H M, 1990, V14, P69, PATIENT MANAGEMENT/RE
E11           1     GARDYNE H, 1995, GEN PRACTITIONER PAR/RE
E12           1     GARDYNER G, 1650, P29, DESCRIPTION NEW WORL/RE

=> s e4-8
L1           16 ("GARDYN J, 1995, V50, P292, AM J HAEMATOL"/RE OR "GARDYN J,
                1995, V50, P292, AM J HEMATOL"/RE OR "GARDYN J, 1995, V50, P292,
                 AMERICAN JOURNAL OF HEMATOLOGY"/RE OR "GARDYN J, 1995, V50,
                P2925, AM J HEMATOL"/RE OR "GARDYN J, 2001, V74, P76, INT J
                HEMATOL"/RE)

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PROCESSING COMPLETED FOR L1
L2            14 DUP REM L1 (2 DUPLICATES REMOVED)
                 ANSWERS '1-5' FROM FILE CAPLUS
                 ANSWERS '6-14' FROM FILE SCISEARCH

=> d bib abs 1-14

L2   ANSWER 1 OF 14  CAPLUS  COPYRIGHT 2003 ACS        DUPLICATE 1
AN   2000:435822  CAPLUS
DN   133:305147
TI   Heparin-induced thrombocytopenia
AU   Depasse, F.; Samama, M. M.
CS   LCL Laboratoire Claude-Levy, Ivry-sur-Seine, 94200, Fr.
SO   Annales de Biologie Clinique (2000), 58(3), 317-326
     CODEN: ABCLAI; ISSN: 0003-3898
PB   John Libbey Eurotext
DT   Journal; General Review
LA   French
AB   A review with 38 refs. Thrombocytopenia is a threatening complication of
     heparin treatment.  The mechanism is the prodn. of antibodies, the most
     frequent target of which is the heparin-platelet factor 4 complex.  These
     antibodies may activate coagulation and lead to venous or arterial
     thromboembolic manifestations.  Clin. features as well as functional and
     immunol. tests are used for diagnosis.  Treatment consists in
     discontinuing heparin administration and using an alternative treatment,
     for which two drugs are indicated in France: Orgaran and Refludan.
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RE.CNT 38 THERE ARE 38 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 14 CAPLUS COPYRIGHT 2003 ACS DUPLICATE 2
AN 2000:90289 CAPLUS
DN 133:28923
TI Broadsheet number 53: activated protein C resistance: diagnosis and clinical management
AU Baker, Ross I.; Eikelboom, John; Street, Alison
CS Clinical Thrombosis Unit, Haematology Department, Royal Perth Hospital, University of Western Australia, Perth, Australia
SO Pathology (1999), 31(4), 365-371
CODEN: PTLGAX; ISSN: 0031-3025
PB Carfax Publishing
DT Journal; General Review
LA English
AB A review with 62 refs. Diseases assocd. with activated protein C resistance, diagnosis, complications and prophylaxis were discussed.
RE.CNT 62 THERE ARE 62 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 3 OF 14 CAPLUS COPYRIGHT 2003 ACS
AN 2000:351385 CAPLUS
DN 132:343317
TI Method of treating heparin-induced thrombocytopenia with protein C
IN Fisher, Charles Jack; Yan, Sau-Chi Betty
PA Eli Lilly and Company, USA
SO PCT Int. Appl., 24 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 2000029014	A1	20000525	WO 1999-US26770	19991110
	W: AE, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, CA, CH, CN, CR, CU, CZ, DE, DK, DM, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, NO, NZ, PL, PT, RO, RU, SD, SE, SG, SI, SK, SL, TJ, TM, TR, TT, TZ, UA, UG, US, UZ, VN, YU, ZA, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM				
	RW: GH, GM, KE, LS, MW, SD, SL, SZ, TZ, UG, ZW, AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE, BF, BJ, CF, CG, CI, CM, GA, GN, GW, ML, MR, NE, SN, TD, TG				
	BR 9915317	A	20010807	BR 1999-15317	19991110
	EP 1128842	A1	20010905	EP 1999-972105	19991110
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
	JP 2002529515	T2	20020910	JP 2000-582060	19991110
	ZA 2001002722	A	20020503	ZA 2001-2722	20010403
	US 2001018050	A1	20010830	US 2001-853859	20010511
PRAI	US 1998-108432P	P	19981113		
	WO 1999-US26770	W	19991110		
	US 1999-439066	A1	19991112		

AB A method is provided for treatment of heparin-induced thrombocytopenia with protein C. The invention provides a needed therapy for a potentially serious and debilitating disorder while avoiding complications such as bleeding tendency, toxicity and general side effects of currently available anticoagulant agents.
RE.CNT 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 4 OF 14 CAPLUS COPYRIGHT 2003 ACS
AN 2000:354147 CAPLUS
DN 133:294335

TI Resistance to activated protein C and inherited thrombosis - molecular mechanism, diagnosis and clinical management
AU Baker, Ross; Eikelboom, John
CS Clinical Thrombosis Unit, Department of Haematology, Royal Perth Hospital, University of Western Australia, Perth, 6001, Australia
SO Advances in Vascular Biology (2000), 6(Platelets, Thrombosis and the Vessel Wall), 307-335
CODEN: AVBIFD; ISSN: 1072-0618
PB Harwood Academic Publishers
DT Journal; General Review
LA English
AB A review, with 194 refs. Studies on the relation between resistance to activated protein C (APC) and inherited thrombosis are reviewed. The recent discovery of APC resistance has substantially changed the understanding of the importance of the protein C anticoagulant pathway in patients with a personal or family history of venous thrombosis. This abnormality is caused by a single point mutation involving the factor V gene which can be detected readily in the clin. lab. by either clotting-based or mol. techniques. The discovery of APC resistance offers an improved understanding of the relation between genetic and environmental factors in the predisposition to venous thrombosis and confirms the polygenic nature of this common clin. problem.
RE.CNT 194 THERE ARE 194 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 5 OF 14 CAPLUS COPYRIGHT 2003 ACS
AN 1999:137387 CAPLUS
DN 130:181172
TI Heparin-induced thrombocytopenia-pathogenesis and treatment
AU Greinacher, Andreas
CS Inst. Immunologie Transfusionsmedizin, Ernst-Moritz-Arndt-Univ., Greifswald, D-17487, Germany
SO Haemostaseologie (Stuttgart) (1999), 19(1), 1-12
CODEN: HAEMD2; ISSN: 0720-9355
PB F. K. Schattauer Verlagsgesellschaft mbH
DT Journal; General Review
LA German
AB A review with 108 refs. is given on pathogenesis and treatment of heparin-induced thrombocytopenia (HIT). Affected patients generate antibodies with specification against complexes of platelet proteins (mainly platelet factor 4) and heparin. The resulting immune-complexes activate platelets via the platelet Fc-receptor and also alterate endothelial cells. This results in massive thrombin generation and activation of the clotting cascade. Therefore, HIT patients have to be further anticoagulated. Hereto mainly 2 drugs are available: the recombinant hirudin Lepirudin and danaparoid-Na. Lepirudin is a direct thrombin inhibitor, danaparoid has mainly anti factor Xa activity. The main treatment options for HIT patients are discussed.
RE.CNT 108 THERE ARE 108 CITED REFERENCES AVAILABLE FOR THIS RECORD
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 6 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 2003:395387 SCISEARCH
GA The Genuine Article (R) Number: 673VY
TI Amyloidosis with IgM monoclonal gammopathies
AU Gertz M A (Reprint); Kyle R A
CS Mayo Clin, Dysproteinemia Clin, 200 1St St Sw, Rochester, MN 55905 USA (Reprint); Mayo Clin, Dysproteinemia Clin, Rochester, MN 55905 USA; Mayo Clin, Dept Lab Med & Pathol, Rochester, MN 55905 USA
CYA USA
SO SEMINARS IN ONCOLOGY, (APR 2003) Vol. 30, No. 2, pp. 325-328.
Publisher: W B SAUNDERS CO, INDEPENDENCE SQUARE WEST CURTIS CENTER, STE 300, PHILADELPHIA, PA 19106-3399 USA.
ISSN: 0093-7754.
DT General Review; Journal

LA English
REC Reference Count: 15

L2 ANSWER 7 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 1999:103605 SCISEARCH
GA The Genuine Article (R) Number: 160TF
TI Thrombotic thrombocytopenic purpura
AU Yeter M (Reprint); Bagcecik G
CS UNIV HACETTEPE, FAC MED, TR-06100 ANKARA, TURKEY (Reprint)
CYA TURKEY
SO AMERICAN JOURNAL OF HEMATOLOGY, (FEB 1999) Vol. 60, No. 2, pp. 171-172.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC, 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0361-8609.
DT Letter; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 4

L2 ANSWER 8 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 1999:204004 SCISEARCH
GA The Genuine Article (R) Number: 173RF
TI Heparin-induced thrombocytopenia: pathophysiology and clinical concerns
AU Greinacher A (Reprint)
CS UNIV GREIFSWALD, INST IMMUNOL & TRANSFUS MED, SAUERBRUCHSTR DIAGNOST ZENTRUM, D-17487 GREIFSWALD, GERMANY (Reprint)
CYA GERMANY
SO BAILLIERES CLINICAL HAEMATOLOGY, (JUN 1998) Vol. 11, No. 2, pp. 461-474.
Publisher: BAILLIERE TINDALL, 24-28 OVAL RD, LONDON NW1 7DX, ENGLAND.
ISSN: 0950-3536.
DT Article; Journal
FS CLIN
LA English
REC Reference Count: 74
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Heparin-induced thrombocytopenia (HIT) is a severe immunological adverse effect of heparin treatment. Recently the pathogenesis of HIT has been resolved regarding the mechanisms of platelet activation, the nature of the most important antigens and the involvement of the clotting cascade. HIT seems to be associated with massive generation of thrombin, which contributes to the thromboembolic complications. Based on these findings, treatment of patients with acute HIT should include cessation of all heparins and further treatment with an anticoagulant with antithrombin activity. Currently, the two most important compounds for further anticoagulation of HIT-patients are danaparoid-sodium and recombinant hirudin.

L2 ANSWER 9 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 1998:509046 SCISEARCH
GA The Genuine Article (R) Number: ZW695
TI Heparin-induced thrombocytopenia and thrombosis syndrome
AU Wallis D E (Reprint); Lewis B E; Messmore H; Wehrmacher W H
CS LOYOLA UNIV, MED CTR, CARDIOVASC INST, BLDG 104, ROOM 3369, 2160 S 1ST AVE, MAYWOOD, IL 60153 (Reprint); LOYOLA UNIV, MED CTR, DEPT MED, SECT HEMATOL, MAYWOOD, IL 60153; HINES VET HOSP, HINES, IL
CYA USA
SO CLINICAL AND APPLIED THROMBOSIS-HEMOSTASIS, (JUL 1998) Vol. 4, No. 3, pp. 160-163.
Publisher: LIPPINCOTT-RAVEN PUBL, 227 EAST WASHINGTON SQ, PHILADELPHIA, PA 19106.
ISSN: 1076-0296.
DT General Review; Journal
FS CLIN
LA English
REC Reference Count: 41

L2 ANSWER 10 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 97:458483 SCISEARCH
GA The Genuine Article (R) Number: XD728
TI Resistance to activated Protein C and factor V Leiden
AU Perry D J (Reprint); Pasi K J
CS ROYAL FREE HOSP, DEPT HAEMATOL, HAEMOPHILIA CTR, POND ST, LONDON NW3 2QQ, ENGLAND (Reprint); ROYAL FREE HOSP, DEPT HAEMATOL, HAEMOSTASIS UNIT, LONDON NW3 2QQ, ENGLAND; UNIV LONDON SCH MED, LONDON, ENGLAND
CYA ENGLAND
SO QJM-MONTHLY JOURNAL OF THE ASSOCIATION OF PHYSICIANS, (JUN 1997) Vol. 90, No. 6, pp. 379-385.
Publisher: OXFORD UNIV PRESS, GREAT CLARENDON ST, OXFORD, ENGLAND OX2 6DP.
ISSN: 0033-5622.
DT General Review; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 44

L2 ANSWER 11 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 97:212562 SCISEARCH
GA The Genuine Article (R) Number: WL892
TI Characterization of the humoral immune response in heparin-induced thrombocytopenia
AU Suh J S; Malik M I; Aster R H; Visentin G P (Reprint)
CS BLOOD CTR SE WISCONSIN INC, BLOOD RES INST, POB 2178, MILWAUKEE, WI 53201 (Reprint); BLOOD CTR SE WISCONSIN INC, BLOOD RES INST, MILWAUKEE, WI 53201; KYUNGPOOK NATL UNIV, SCH MED, DEPT CLIN PATHOL, TAEGU 702701, SOUTH KOREA; MED COLL WISCONSIN, DEPT MED, MILWAUKEE, WI 53226; MED COLL WISCONSIN, DEPT PATHOL, MILWAUKEE, WI 53226
CYA USA; SOUTH KOREA
SO AMERICAN JOURNAL OF HEMATOLOGY, (MAR 1997) Vol. 54, No. 3, pp. 196-201.
Publisher: WILEY-LISS, DIV JOHN WILEY & SONS INC 605 THIRD AVE, NEW YORK, NY 10158-0012.
ISSN: 0361-8609.
DT Article; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 24
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Recent reports indicate that antibodies associated with heparin-induced thrombocytopenia and thrombosis (HITP) are specific for complexes formed between heparin and the heparin-binding, platelet alpha granule protein, platelet factor 4 (PF4). As with other disorders mediated by immune complexes (IC), the characteristics of the involved immunoglobulins could affect the ability of IC to cause symptoms. We therefore studied the class, subclass, and potency of antibodies specific for heparin:PF4 complexes formed by two groups of patients: one with severe thrombocytopenia, with or without thrombosis, and a positive serotonin release assay (SRA) (Group 1) and another with mild or absent thrombocytopenia, absence of thrombosis, and a negative SRA despite having formed antibodies reactive with heparin:PF4 complexes (Group 2).

IgG antibodies were more common in the Group 1 patients (100%) than in Group 2 (46%), whereas IgM antibodies were more common in Group 2 (81%) than in Group 1 (42%) (P = 0.009). About half of each group formed IgA antibodies. In each group, the IgG antibodies were predominantly IgG1 (82%); 42% were IgG3. Only one IgG2 antibody was identified in a total of 52 antibody formers. Antibodies of the IgG class were consistently of higher titer in Group 1 patients than in Group 2 patients (P < 0.001).

Recent reports suggest that the H131 form of the Fc gamma RII receptor, which binds preferentially to lgG2 Pc, is found with greater than expected frequency in patients with HITP. Identification of only one IgG2 antibody among 38 antibodies of the IgG class argues against a unique role for antibodies of this subclass in the pathogenesis of HITP. The finding that titers of antibodies in Group 1 patients were a significantly higher titer

than in Group 2 patients suggests that development of the full-blown HITP syndrome may require the formation of antibodies of unusually high titer. (C) 1997 Wiley-Liss, Inc.

L2 ANSWER 12 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 97:154852 SCISEARCH
GA The Genuine Article (R) Number: WH486
TI Activated protein C resistance due to a common factor V gene mutation is a major risk factor for venous thrombosis
AU Zoller B (Reprint); Hillarp A; Berntorp E; Dahlback B
CS UNIV LUND HOSP, DEPT CLIN CHEM, MALMO, SWEDEN (Reprint); UNIV LUND HOSP, DEPT COAGULAT DISORDERS, MALMO, SWEDEN
CYA SWEDEN
SO ANNUAL REVIEW OF MEDICINE, (MAY 1997) Vol. 48, pp. 45-58.
Publisher: ANNUAL REVIEWS INC, 4139 EL CAMINO WAY, PO BOX 10139, PALO ALTO, CA 94303-0139.
ISSN: 0066-4219.
DT General Review; Journal
FS LIFE; CLIN
LA English
REC Reference Count: 103
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB Inherited resistance to activated protein C (APC) was recently discovered to be a cause of familial thrombophilia and is now known to be the most common genetic risk factor for venous thrombosis. It is caused by a single point mutation in the gene for factor V, which predicts substitution of arginine (R) at position 506 with a glutamine (Q). Accordingly, the activated form of mutated factor V (FVa:Q(506)) is more slowly degraded by activated protein C than normal FVa (FVa:R(506)) is, resulting in hypercoagulability and a lifelong 5- to 10-fold increased risk of venous thrombosis. Previously known inherited hypercoagulable states, i.e. deficiencies of the anticoagulant proteins antithrombin III, protein S, and protein C, are found in fewer than 10-15% of thrombosis patients in western countries, whereas inherited APC resistance is present in 20-60% of such patients. The FV mutation is common in populations of Caucasian origin, with prevalences ranging from 1-15%, whereas it is not found in certain other ethnic groups such as Japanese and Chinese. The high prevalence of APC resistance, in combination with the availability of simple laboratory tests, will have a profound influence on the development of therapeutic and prophylactic regimens for thrombosis and will, it is hoped, result in a decreased incidence of thromboembolic events.

L2 ANSWER 13 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 96:360614 SCISEARCH
GA The Genuine Article (R) Number: UJ105
TI HEPARIN-INDUCED THROMBOCYTOPENIA - UNDERSTANDING IMPROVES BUT QUESTIONS REMAIN
AU ASTER R H (Reprint)
CS MED COLL WISCONSIN, MADISON, WI, 00000 (Reprint)
CYA USA
SO JOURNAL OF LABORATORY AND CLINICAL MEDICINE, (MAY 1996) Vol. 127, No. 5, pp. 418-419.
ISSN: 0022-2143.
DT Editorial; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 16

L2 ANSWER 14 OF 14 SCISEARCH COPYRIGHT 2003 THOMSON ISI
AN 96:777564 SCISEARCH
GA The Genuine Article (R) Number: VN014
TI PATIENTS TREATED WITH UNFRACTIONATED HEPARIN DURING OPEN-HEART-SURGERY ARE AT HIGH-RISK TO FORM ANTIBODIES REACTIVE WITH HEPARIN-PLATELET FACTOR 4 COMPLEXES
AU VISENTIN G P (Reprint); MALIK M; CYGANIAK K A; ASTER R H

CS BLOOD CTR SE WISCONSIN INC, BLOOD RES INST, POB 2178, MILWAUKEE, WI, 53201 (Reprint); MED COLL WISCONSIN, DEPT MED, MILWAUKEE, WI, 53226; MED COLL WISCONSIN, DEPT PATHOL, MILWAUKEE, WI, 53226
CYA USA
SO JOURNAL OF LABORATORY AND CLINICAL MEDICINE, (OCT 1996) Vol. 128, No. 4, pp. 376-383.
ISSN: 0022-2143.
DT Article; Journal
FS LIFE; CLIN
LA ENGLISH
REC Reference Count: 20
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Recent studies have demonstrated a strong association between type II (immunologically mediated) heparin-induced thrombocytopenla/thrombosis (HITP) and antibodies reactive with complexes consisting of heparin and platelet factor 4 (PF4), a heparin-binding protein normally found in platelet-or granules. However, the frequency with which such antibodies develop in patients given treatment with heparin has not yet been defined. We studied the development of heparin:Pf4-specific antibodies in 51 patients who received a single dose of unfractionated heparin (UFH) during cardiac catheterization and were then given UFH or low-molecular-weight heparin (LMWH) again during and after open heart surgery. Eleven of the 51 patients (22%) had antibodies reactive with heparin:PN when they were admitted for cardiac surgery; these antibodies were mainly of the Immunoglobulin M (IgM) class and were apparently stimulated by exposure to UFH at cardiac catheterization. Seventeen of 34 patients (50%) without preexisting antibody who were given UFH during and for 1 to 3 days offer surgery formed immunoglobulin G antibodies or IgM antibodies (or both) by the sixth postoperative day. Overall, 27 of 44 patients (61%) who were given UFH at surgery had antibodies by the time of hospital discharge. None of 6 patients without preexisting antibody who were given LMWH at surgery formed antibodies ($p < 0.03$). However, LMWH was given as a single injection only on the day of surgery. The titer of the antibodies formed by patients receiving UFH ranged from 1:10 to 1:200, significantly lower than those in patients with a clinical diagnosis of HITP. Moderate thrombocytopenia was common after open heart surgery, but platelet levels in patients who had preexisting antibodies or formed new antibodies did not differ significantly from those in patients without antibody. Clinically significant thrombosis did not develop in any patient and HITP was not diagnosed in any patient. Antibodies reactive with heparin:PF4 formed in only 3 of 66 patients (4.5%) undergoing other types of surgery, One of these patients had been given UFH 3 months previously; the other 2 may have been exposed to heparin used to flush intravenous lines postoperatively, No antibodies reactive with heparin:PF4 were found in any of 108 normal subjects. We conclude that UFH is more immunogenic than has been thought and that patients exposed to this anticoagulant during open heart surgery are at high risk to form low titer (less than or equal to 1:200) antibodies reactive with heparin:PM. Further studies are needed to determine whether such antibodies are clinically significant-that is, whether sensitized patients are at risk to develop HITP if heparin treatment is continued for more than I to 3 days or is reinstituted at a later date.

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FULL ESTIMATED COST	63.66	63.87

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